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Disrupting the experience of control in the human brain: pre-supplementary motor area contributes to the sense of agency

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Title: Disrupting the experience of control in the human brain: pre-Supplementary
Motor Area contributes to the sense of agency

Running head: Experience of control in the human brain

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Abstract

The feeling of controlling events through one's actions is fundamental to human experience, but its neural basis remains unclear. This "sense of agency" can be measured quantitatively as a temporal linkage between voluntary actions and their external effects. We investigated the brain areas underlying this aspect of action awareness by using theta-burst stimulation to locally and reversibly disrupt human brain function. Disruption of the pre-Supplementary Motor Area (a key structure for preparation and initiation of voluntary action) was shown to reduce the temporal linkage between a voluntary keypress action and a subsequent electrocutaneous stimulus. In contrast, disruption of sensorimotor cortex, which processes signals more directly related to action execution and sensory feedback, had no significant effect. Our results provide the first direct evidence of a pre-SMA contribution to sense of agency.

Key words: Voluntary action; Agency; Pre-Supplementary Motor Area; Sensorimotor cortex; Theta-burst stimulation; Transcranial magnetic stimulation

Introduction

The ability to plan and control actions in order to achieve desired goals is a hallmark of human and animal intelligence. In humans, at least, such operant actions also produce a characteristic conscious experience of controlling external events, called “sense of agency” (SoA).

Several studies have sought to identify the brain circuits underlying SoA. Most have manipulated the sensory feedback associated with action, and asked subjects to explicitly judge whether they are the agent responsible for the feedback or not. The inferior parietal cortex is activated in these explicit attributions of agency (e.g. Chaminade & Decety, 2002; Farrer & Frith, 2002; Farrer et al., 2008). Moreover, patients with damage to the parietal cortex perform poorly on tasks where they are asked to judge if visual feedback corresponds to their own action or not (Sirigu, Daprati, Pradat-Diehl, Franck, & Jeannerod, 1999). However, parietal regions seem to be concerned more with non-agency in cases of incongruent feedback (“that was *not* me”) than with the positive experience of agency itself. Furthermore the explicit judgements of agency used in such tasks may involve different processes from the background feeling, or sense, of agency that accompanies our normal voluntary action (Synofzik, Vosgerau, & Newen, 2008).

Implicit measures provide an alternative way of quantifying SoA, and may be better suited to capturing this background feeling about one’s own action (Synofzik et al., 2008). Such measures are implicit in that participants do not make explicit attributions or judgements of agency. A number of implicit measures of the SoA have been proposed, including the attenuation of self-produced sensations (Blakemore, Wolpert,

& Frith, 2000) and changes of time perception associated with voluntary movement. Here we focus on the latter.

When a voluntary action is followed by a sensory effect, there is subjective compression of the interval between the two events. In one paradigm, participants are asked to estimate the time of the action, or the time of a subsequent tone, in separate blocks (Haggard, Clark, & Kalogeras, 2002). Actions are perceived as shifted later in time towards the tone, in comparison to a baseline condition in which subjects' actions do not produce tones. A tone that follows an action is perceived as shifted earlier towards the action that causes it, relative to a baseline condition involving tones but no actions (Haggard et al., 2002; Tsakiris & Haggard, 2003). These shifts were found for voluntary actions but not for involuntary movements (Haggard et al., 2002) or pairings of two sensory events (Haggard, Aschersleben, Prinz, & Gehrke, 2002; Stetson, Cui, Montague, & Eagleman, 2006) suggesting a mechanism specific to intentional action. A similar compression is found if participants judge the interval between action and tone, rather than the time of each event individually (Engbert, Wohlschläger, Thomas, & Haggard, 2007; Engbert, Wohlschläger, & Haggard, 2008).

We have investigated the neural substrates involved in sense of agency by measuring the effects of locally disrupting brain activity on this “intentional binding” effect. We used non-invasive continuous theta-burst brain stimulation (cTBS), a protocol that has consistently been shown to inhibit neural activity in the target brain region (Huang & Rothwell, 2004; Huang, Edwards, Rounis, Bhatia, & Rothwell, 2005; Huang, Chen, Rothwell, & Wen, 2007). Two candidate brain regions were selected: The sensorimotor hand area (SMHA), and the pre-Supplementary motor area (pre-SMA).

The SMHA is primarily concerned with motor execution and sensorimotor feedback (e.g. Weiller et al., 1996), while the pre-SMA is involved in the more cognitive aspects of internal movement generation (Picard & Strick, 2001), and with the conscious urge to act (Fried et al., 1991). In this way, both target regions are plausibly neural substrates of SoA, but are distinguishable in terms of both their function and time of contribution. Thus, any change in the intentional binding effect resulting from disrupting either one of these areas would help to identify what neural signals may underlie SoA. These disruptions can be compared to the effects of stimulation of a control site not likely to be involved in SoA, but generating the same non-specific effects of TMS, such as arousal, auditory and cutaneous stimulation. If cTBS over either pre-SMA or SMHA significantly alters binding, we may infer that the relevant brain area contributes to SoA. Further, if cTBS at both sites significantly alters binding we may compare the deficits across the two stimulation sites to establish the relative contributions of each. However, this analysis would make sense only after showing that both areas contribute to binding.

Materials and Methods

Participants

10 right-handed individuals (5 females; mean age 25.20 years) took part with ethics committee approval, in three sessions on separate days. Participants were screened before entry into the experiment to ensure they showed an intentional binding effect (Haggard et al., 2002). Of 20 participants initially screened, 16 showed significant binding for actions and 17 showed significant binding for effects. We selected the 13 participants who showed significant binding for *both* actions and effects (see

Procedure: Behavioural test for details of the task). 10 of these 13 participants were

recruited in the present experiment (3 were unable to attend follow-up testing). The logic behind this screening is as follows: intervention to disrupt a behavioural effect can only sensibly be studied when the effect is present. Since these 10 individuals showed evidence of intentional binding, they were particularly appropriate participants in our attempt to disrupt intentional binding. By implication our results apply to the population of participants showing intentional binding, which may not be representative of the population as a whole. Similar screening has been used in other neurophysiological studies of motor representation (e.g. Wolters et al., 2003; Stefan et al., 2006). The presence of the binding effect within a wider, unscreened population was established in previous studies (Haggard et al., 2002).

Procedure: Behavioural test

Behavioural testing occurred immediately after cTBS (see *Neurophysiological methods* below for details), and followed previous methods (e.g. Haggard et al., 2002; see also Figure 1A for set-up). Participants were instructed to press a key with their right index finger at a time of their choosing. This caused a cutaneous somatosensory stimulus (a mild shock) to the right little finger 250 ms later in agency conditions, but not in baseline conditions. Whereas previous studies used an auditory tone as the effect of action (e.g. Moore & Haggard, 2008), we chose to use a somatosensory stimulus in this case, so that the key signals for both action and for the effects of action would be co-localised in a single brain area (the sensorimotor hand area, SMHA). This increased the likelihood of disrupting neural processing of both action and effect signals.

While making actions and receiving shocks, participants viewed a clock hand rotating with a period of 2560 ms. Following the action and shock delivery, the clock hand continued rotating for a random period of time. After the clock stopped the participant verbally reported the position of the clock hand at which they pressed the key, or the time at which they felt the shock. Action judgements and shock judgements were performed in separate blocks of trials. In two further baseline blocks, participants made actions but never received shocks, or received shocks at a random time, but never made actions. Each block contained 20 trials. Block order was randomised anew for each session (see below). Testing lasted approximately 15 minutes, well within the window of cTBS efficacy (Huang et al., 2005).

Prior to each session, each participant's detection threshold for cutaneous shocks from ring electrodes on the right little finger (one ring electrode placed on the proximal phalanx and the other on the middle phalanx) was identified by an ascending staircase procedure. Shock intensity was varied by changing pulse width until the shock was detected, then decreased with half the step size until the shock was missed, then increased again with further halving of the step size, until the fourth reversal of responding (Levitt, 1971). The average of the final two reversals was taken as the detection threshold. The experimental shocks used 140% of each participant's detection threshold for that session.

Figure 1 about here

Neurophysiological methods

Subjects sat comfortably in a reclining chair. Surface-electromyography (EMG) was recorded from the first dorsal interosseous (FDI) muscle of the right hand using 9 mm diameter Ag-AgCl surface electrodes in a belly-tendon montage. The active electrode was placed over the belly muscle, the reference electrode over the metacarpophalangeal joint of the index finger. Responses were amplified, filtered with a 20 Hz-2 kHz passband, and digitally sampled at 5 kHz.

A Super Rapid Magstim package (Magstim Co, Whitland, Dyfed, UK) was connected to a 70 mm figure-of-eight coil. To stimulate primary motor cortex the coil was placed tangentially to the skull with the handle pointing backwards and rotated 45° away from the midline. The optimal coil position ('hotspot') for activating the contralateral FDI was determined as the site where stimulation at a slightly suprathreshold stimulus intensity consistently produced the largest Motor Evoked Potential (MEP). We then calculated the Active Motor threshold (AMT) at this hotspot, by asking the participant to maintain a slight isometric contraction (5-10% of maximum voluntary contraction), and identifying the lowest stimulus intensity which elicited a mean MEP > 200µV from at least five of ten consecutive trials (Rossini et al., 1994).

Thetaburst stimulation (TBS) consists of repeating bursts of TMS, with 3 pulses at 50 Hz repeated at 200 ms intervals (i.e., at 5 Hz). A train of 600 pulses, termed continuous TBS (cTBS), can suppress local cortical excitability for approximately 20-30 minutes (Huang et al., 2005). Stimulation intensity was set to 80 % of the AMT for the FDI muscle at the motor cortex hot spot. All participants had cTBS stimulation at three stimulation sites (sensorimotor hand area (SMHA), pre-SMA, S1-leg area) in

separate sessions in a random order. Participants were not informed of the specific stimulation site at each session. No adverse consequences of cTBS occurred. For SMHA stimulation the optimal stimulation site to evoke MEPs in the FDI muscle was used. To target pre-SMA a site 4 cm anterior to the hotspot of the FDI was marked in the sagittal midline (Cz of the international 10-20-EEG system) of the scalp (Rushworth, Hadland, Paus, & Sipila, 2002; Mars et al., 2009). In one participant, bilateral twitches in the leg muscles were evoked at this location by high stimulation intensity (90% maximal stimulator output). The coil was then moved 0.5 cm more anteriorly so that twitches no longer occurred. To target the S1-leg area as a control site, a position 2 cm posterior to the hot spot for the FDI muscle in the sagittal midline of the skull was marked on the scalp.

The location of the scalp stimulation sites with respect to underlying brain anatomy was examined with TMS-MRI co-registration in a separate group of 3 subjects. The three stimulation positions used in the main experiment were identified in the same way as for the experimental subjects, and marked on each subject's scalp. A high resolution three-dimensional volumetric structural magnetic resonance image (MRI) was obtained for each subject. The cortical surface was displayed as a three-dimensional representation using Brainsight Frameless Stereotaxic software (Rogue Research, Montreal, Quebec, Canada). Each stimulation site was identified on the MRI image using a pointer tool (Brainsight Frameless P-697), while the position of the pointer and the subject's head were monitored using a Polaris Optical Tracking system (Northern Digital, Waterloo, Ontario, Canada). One example is shown in Figure 1B. The anatomical location of the pre-SMA was identified from the border between the SMA proper and the pre-SMA which is defined by the plane

perpendicular to the anterior commissure-posterior commissure (AC-PC) line at the level of the AC (Picard and Strick, 1996). We could confirm in all 3 subjects that the site of TMS stimulation on the scalp lay over the anatomically-defined pre-SMA in the brain.

The order of testing sessions was counterbalanced using the Latin square procedure. All three of each participants' testing sessions took place at a similar time of day (either am or pm), to minimise circadian effects. The order of perceptual judgement blocks was randomised anew for each session.

Data Analysis

The perceived time of action or shock on each trial was compared to actual onset time, and a mean temporal error calculated for each block. The mean error for actions and shocks in baseline blocks was subtracted from that in experimental blocks. Subtracting these baseline estimates allowed us to calculate the shift in the perceived time of the action when the shock was present, and the shift in the perceived time of the shock when caused by the action. These shifts serve as measures of 'intentional binding' between action and effect. By convention a positive temporal shift indicates delayed awareness. Therefore, a positive shift for actions represents binding of actions towards shocks, while a negative shift for shocks represents binding of shocks towards the preceding action. Finally, an overall measure of binding between action and effect was calculated as action binding minus tone binding. Larger values of this measure indicate stronger action-effect binding. All data are presented in

Supplemental Table 1.

Controlling for possible cTBS-induced changes in sensory intensity

CTBS could, in principle, alter somatosensory function, leading to changes in the perceived time of shock stimuli. To control for this possibility, we additionally checked whether cTBS affected perceived shock intensity, which could in turn affect timing judgements.

Therefore, before each cTBS session, subjects received 5 ‘reference shocks’, triggered by their own keypress, and at the same intensity as in the main experiment. They were asked to attend to the intensity of these reference shocks for subsequent comparison. After each block of trials (except the baseline action condition in which there were no shocks), participants were asked to compare the shock intensity they experienced during the block with the initial reference shocks, using a 7 point scale (-3 = experimental shocks much less intense than reference, +3 = experimental shocks much more intense). These ratings were averaged across the blocks in each, to give a total measure of perceived shock intensity following stimulation at each TMS site (see **Supplemental Table 1**).

Results

The experience of intentionally controlling shocks changed the perceived time of both action and shock. Overall binding between action and effect is measured by the action shift minus the effect shift (Figure 2). If SoA depends on brain circuits for internal generation of action, then overall binding following pre-SMA stimulation should be reduced relative to control stimulation. Alternatively, if SoA depends on brain circuits for action execution and somatosensory feedback, then overall binding following SMHA stimulation should be reduced relative to control. Because both

areas might make independent contributions, we made no prediction about the difference between pre-SMA and SMHA stimulations.

Pre-SMA stimulation indeed reduced the overall amount of binding (118 ms \pm 22 ms SE) relative to control stimulation of the sensory leg area (153 ms \pm 24 ms SE) (2-tailed paired samples t-test: $t(9) = 3.49$, $p = .007$). Testing each shift separately showed that pre-SMA stimulation influenced the shift in perceived time of the effect (2-tailed paired samples t-test: $t(9) = 2.32$, $p = .045$) but not the shift in action (2-tailed paired samples t-test: $t(9) = .058$, $p = .960$). Interestingly, the effect of stimulation on our composite binding measure is statistically stronger than that on action binding or effect binding individually. This pattern was found because pre-SMA stimulation influenced primarily action binding for some subjects, and effect binding for others. However, the implied interval between action and effect was influenced with much greater reliability than either event individually.

SMHA stimulation had a much smaller influence on overall binding (137 ms \pm 31 ms SE). Moreover, this influence was rather variable across individuals, and did not differ significantly from control stimulation over the sensory leg area (2-tailed paired samples t-test: $t(9) = .469$, $p = .650$). Testing each shift separately revealed that SMHA stimulation did not significantly affect either action binding (2-tailed paired samples t-test: $t(9) = .119$, $p = .911$) or effect binding (2-tailed paired samples t-test: $t(9) = .758$, $p = .468$). Since we found no reliable evidence that the SMHA was involved binding, we could not proceed to directly compare the magnitudes of the two disruptions (see Introduction).

We also investigated whether cTBS might affect time perception generally, by comparing baseline judgements across the different stimulation conditions. Baseline judgements of both actions and effects were unaffected by pre-SMA stimulation relative to control site (2-tailed paired samples t-test – baseline action: $t(9) = .095$, $p = .926$; baseline tone: $t(9) = .710$, $p = .496$). Equally, SMHA stimulation produced no change in baseline judgements relative to control site (2-tailed paired samples t-test – baseline action: $t(9) = .582$, $p = .575$; baseline tone: $t(9) = .246$, $p = .811$). Therefore, it seems unlikely that our results are merely an artefact of poor time perception, or similar non-specific factors.

Figure 2 about here

Finally, we investigated whether cTBS might have affected perceived shock intensity, perhaps leading to differences in perceived timing. We found no differences in perceived intensity between control (S1-leg area) and pre-SMA stimulation (2-tailed paired samples t-test: $t(9) = .483$, $p = .641$), nor between control and SMHA stimulation (2-tailed paired samples t-test: $t(9) = 1.41$, $p = .193$) (see **Supplemental Table 1** for data). This suggests that TMS-induced changes in perceptual timing are unlikely to be due to changes in perceived intensity.

Discussion

In this study, we used cTBS to shed light on the neural substrate of SoA. Two candidate brain regions were stimulated: pre-SMA and SMHA. The effects of stimulating each area on intentional binding (a temporal measure of SoA) were compared with stimulation of a control area (sensory leg area). Following pre-SMA

disruption, we found significant reduction in overall intentional binding, and particularly in the binding of sensory consequences towards actions. Disruption of SMHA with cTBS did not significantly reduce binding relative to control area.

Limitations

We have used an indirect measure of agency, based on time perception. In contrast, previous studies investigated explicit judgements of agency (Farrer & Frith, 2002). Intentional binding is a useful measure of sense of agency to the extent that it occurs only in situations that indeed involve agency, i.e. when a self-generated movement produces a sensory effect, but not when involuntary movements lead to the same effects (Haggard et al., 2002), or where two stimuli co-occur without voluntary action (Haggard, Aschersleben, Prinz, & Gehrke, 2002; Stetson, Cui, Montague, & Eagleman, 2006). However, it remains unclear whether intentional binding, and sense of agency, *always* accompanies *all* self-generated movements. Several studies suggest that binding (Moore, Wegner & Haggard, 2009; Ebert & Wegner, 2010) and action experience in general (Haggard, Cartledge, Dafydd, & Oakley, 2004) can be influenced by contextual factors and suggestions about one's action.

Many studies of voluntary action, including this one, have focussed on the pre-SMA (Fried et al., 1991). Our pre-SMA localisation followed that used successfully in previous studies (Rushworth et al., 2002; Mars et al., 2009), and was confirmed by neuronavigation in a separate group of three participants. However, a potential limitation of our study is the absence of any functional localiser confirming that our cTBS was located over pre-SMA. Therefore, our cTBS could also have affected neighbouring regions, such as SMA-proper, so disruption there might underlie our

effects. However, the relatively low stimulation intensity of 80% Active Motor Threshold makes a contamination of other areas by current spread rather unlikely. Co-registration of our pre-SMA stimulation site, albeit in a different group of subjects, also confirmed that the coil was located anterior to SMA-proper which was defined anatomically as being anterior to the plane perpendicular to the AC-PC-line at the level of the AC.

A related concern is the possible remote effects of TMS (Bestmann, Baudewig, Siebner, Rothwell, & Frahm, 2004). In principle, our pre-SMA results could arise from disruption to regions connected to pre-SMA rather than in pre-SMA itself. This possibility cannot be ruled out completely. The candidate areas for such remote effects should be strongly connected to the pre-SMA. The major input to the pre-SMA comes from the basal ganglia (Akkal, Dum, & Strick, 2007). The major targets of pre-SMA output are prefrontal regions (Luppino, Matelli, Camarda, & Rizzolatti, 1993; Bates & Goldman-Rakic, 1993; Johansen-Berg et al., 2004), and the basal ganglia (Inase, Tokuno, Nambu, Akazawa, & Takada, 1999; Lehericy et al., 2004). However, it is unclear whether disruption of these remote areas would produce changes in SoA. A candidate prefrontal region with respect to SoA is the dorsolateral prefrontal cortex (DLPFC). The current literature gives few indications whether remote disruption of these areas could be the basis of our findings. Early imaging studies showed that DLPFC activity is greater for active vs. passive movements (e.g. Frith, Friston, Liddle, & Frackowiak, 1991), which suggested a role in the initiation of voluntary action. However, more recent imaging research suggests that the specific function of DLPFC is in the selection between action alternatives, particularly under conflict, rather than volition per se (Rowe, Toni, Josephs, Frackowiak, & Passingham, 2000).

In our study, participants made only one action, so action selection was absent. This reduces the putative influence of DLPFC. The basal ganglia (BG) are another possible source of remote effects of pre-SMA stimulation. Here it is harder to rule out remote effects. One recent study showed *intact* intentional binding in patients with Parkinson's Disease (Moore et al., 2010). While the effects on the BG of Parkinson's Disease of pre-SMA cTBS are not directly comparable, this result at least suggests that remote BG disruption is unlikely to explain our pre-SMA findings.

The pre-SMA: a premotor contribution to SoA?

The pre-SMA plays a key role in the initiation and control of voluntary action (Deiber et al., 1991; Hikosaka et al., 1996; Nachev, Wydell, O'Neill, Husain, & Kennard, 2007). Higher order features of motor cognition have also been attributed to the pre-SMA; direct stimulation of this region produces a conscious 'urge to move' (Fried et al., 1991), and attending to the intention to move activates this area (Lau, Rogers, Haggard, & Passingham, 2004). Taken together these results suggest that the pre-SMA plays a key role in the experience of volition, by coding the conscious intention to move. Our results suggesting that pre-SMA may also contribute to SoA. Lasting disruption of putative pre-SMA significantly weakened a temporal marker of agency, namely the perceived association between actions and effects.

Theoretically, SoA could arise from either of two distinct processes. On the one hand, SoA may involve a prediction - based on the neural commands for action - that the sensory effect will occur. On the other hand, the brain might infer, or *postdict* from the conjunction of action and effect (Hume, 1739) that the action caused the effect, as in illusions of conscious will (Wegner, 2002). Recent behavioural studies

show that the intentional binding effect involves a combination of both processes. First, the binding of outcomes towards actions was significantly reduced in blocks where additional outcome events could sometimes occur in the absence of action, compared to blocks where outcomes occurred only directly after actions. This suggests that outcome binding reflects an inference based on knowledge about the distribution of outcomes (Moore, Lagnado, Deal, & Haggard, 2009). Second, when the outcome following an action was occasionally omitted, a shift in perceived time of action towards the predicted tone was nevertheless observed (Moore & Haggard, 2008). When *both* predictive and postdictive information are available, they may be combined in a flexible way, according to the availability and reliability of each (Moore, Wegner, & Haggard, 2009).

Distinguishing between predictive and postdictive components of SoA requires varying the conditional probabilities of tones and actions. In the present study, actions were always followed by shocks in the experimental blocks so both prediction and postdiction could have contributed to binding. Therefore, we cannot conclusively determine whether the reduced binding effect caused by pre-SMA stimulation reflects a disruption of prediction or of postdiction. However, a predictive role is more consistent with other evidence regarding the role of pre-SMA in action preparation. In particular, recordings from pre-SMA have demonstrated its role both in action preparation and in anticipatory processing of warning signals prior to action (Ikeda et al., 1999).

We found that pre-SMA cTBS disrupted effect binding more than action binding. Recent computational models also emphasise the role of effect prediction in SoA

(Blakemore, Wolpert, & Frith, 2002) Specifically, pre-SMA may link intentions to the sensory consequences of the intended action, a view which is consistent with ideomotor theories of voluntary action (James, 1890). A previous study found that disruption of this area abolished sensory suppression during voluntary movement (Haggard & Whitford, 2004). In the present study, we showed analogous effects on the linkage between action and effect. Disruption of the pre-SMA reduced the tendency to link representations of action and effect across time. This result suggests that the pre-SMA may use motor information to generate predictions of the sensory consequences of action.

Care is needed in interpreting our SMHA results. First, there was some evidence of reduced binding following SMHA stimulation, though this was not statistically reliable. In particular, effects of SMHA stimulation were highly variable across individuals. This inter-individual variability presumably did not merely reflect poor localisation of SMHA, since objective physiological criteria were used to localise this area in each participant. Because we did not find strong evidence for any SMHA role in binding, we could not sensibly compare the contributions of pre-SMA and SMHA to investigate which was more important. The absence of any SMHA effect in our data could be a simple statistical power limitation, given our relatively small number of participants. More research is required to confirm whether SMHA contributes to binding or not.

In conclusion, we show that the subjective feeling of control, as indexed by the temporal linkage between actions and effects, depends at least partly on the pre-SMA. More specifically, we suggest that the pre-SMA makes a special contribution to SoA,

housing the predictive mechanisms contributing to the SoA. These results shed light on the neural substrate of SoA, and suggest a strong link between SoA, motor prediction, and medial premotor cortex.

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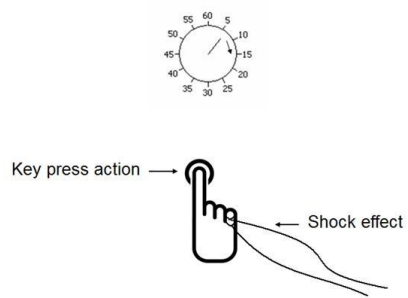
Figure Captions

Figure 1. A) Experimental set-up used in the study. See text for details. B) Putative sites of cTBS stimulation (based on neuro-navigation in a separate group of three participants).

Figure 2. A) Upper row: In experimental conditions, actions were followed by shocks 250 ms later. Lower 3 rows: changes in the perceived time of actions and effects following cTBS in each condition. Black circles indicate the mean perceived time of actions, dashed vertical lines indicate the baseline judgements of actions in that session. Grey triangles indicate the mean perceived time of shocks, dashed vertical lines indicate baseline shock judgements in that session. Numbers indicate the size of the shift in ms. Note the reduced binding following preSMA cTBS. B) Mean overall binding between action and effect (action shift minus shock shift) in ms for each site of stimulation. Bars represent Standard Error of the mean across participants. Asterisks denote significant difference ($p < .01$)

Figure 1

A)



B)

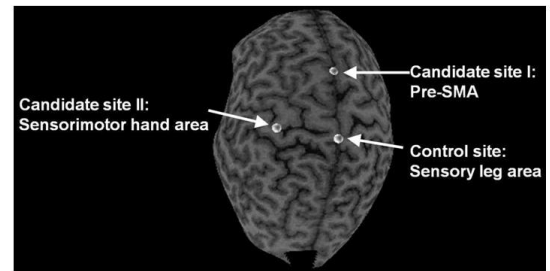


Figure 2

